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13. ABSTRACT *(Maximum 200 words)*

Several general principles are emerging from these studies. First, the functional organization of neural circuits is dynamic, and a single circuit, such as a CPG, can produce several distinct outputs, which in turn, can mediate different behaviors. Second, modulatory transmitters can regulate the functional organization of circuits as well as their responsiveness to inputs. Third, motivational systems can influence behaviors, in part, by acting on motor systems, such as CPGs. Fourth, motor systems possess cellular mechanisms capable of supporting complex forms of neuronal plasticity, which in turn, may contribute to learning and memory. These general principles illustrate that motor behaviors are governed by highly adaptive neural networks and help to explain how systems of nerve cells function to produce and modulate behavior.

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Final Technical Report

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I. Objectives

There were no changes in the objectives of the project.

II. Status of Effort

The overall goal of the project was to analyze the cellular, synaptic and network processes that underlie the genesis and adaptive control of rhythmic neural activity. The investigation focused on an ensemble of identified neurons that functions as an ultradian oscillator. Progress was made in three areas. First, the rhythmic activity in output pathways of the oscillator were characterized. Several distinct patterns were identified, which in turn represented neural correlates of different behaviors. Second, additional cellular and synaptic elements of the oscillator were identified and a 'wiring diagram' of the oscillator was developed. Third, the adaptive responses of the oscillator to various stimuli were characterized. The results indicated that the functional configuration of the oscillator could be reprogrammed by factors such as the motivational state of the animal, modulatory transmitters and highly specific temporal patterns of sensory simulation. Several general principles emerged from these studies. First, the functional organization of neural oscillators is dynamic, and a single oscillator can mediate several temporal programs. Second, the adaptive control of the oscillator is widely distributed among higher-order processes (e.g., motivational states), sensory systems and possibly other oscillatory circuits. Third, properly timed input signals can induce relatively long-lasting changes in the temporal patterns of neural activity generated by an ultradian oscillator.

III. Accomplishments/New Findings

The overall objective of AFOSR sponsored research in this laboratory is to analyze the properties of identified neurons and neural circuits underlying adaptive behavior. During previous funding periods, investigations focused primarily on elements of a neural circuit that mediates a defensive withdrawal reflex in the marine mollusk *Aplysia*. During the current funding period, however, investigations focused on an neural circuit that generates complex patterns of rhythmic neural activity. This rhythmic activity, in turn, mediates aspects of consummatory feeding in *Aplysia*. A brief summary of the progress made during the current funding period follows the list of publications.

A. Characterization of Rhythmic Activity in Output Pathways of the CPG and Responses of the CPG to Non-Specific Stimulation of Input Pathways

Consummatory feeding behaviors of *Aplysia* are a series of intricate rhythmic movements involving coordination among structures of the mouth (lips and jaws) and foregut (buccal mass, radula and esophagus). As a functional group, neural circuits that initiate and maintain such behavioral rhythms are referred to as central pattern generators (CPGs). The CPG that mediates rhythmic movements during feeding is located in the buccal ganglia. By using a variety of intact and semi-intact preparations, considerable progress has been made in defining the patterns of rhythmic neural activity that emanate from the buccal CPG the various consummatory behaviors. The extent to which these patterns of neural activity are manifest *in vitro* had not been fully established, however. During the current funding period, this issue was address by monitoring rhythmic activity in isolated buccal ganglia and comparing these patterns to those previous recorded *in vivo*.

Figure 1 illustrates an extracellular recording of spontaneous rhythmic neural activity that was generated in an experimental preparation consisting of the isolated buccal ganglia. The extracellular records revealed a complex pattern of rhythmic neural activity that was characterized by bursts of large-unit activity in all four buccal nerves. The phase relationships among the spontaneous bursts of activity in each nerve recording was analyzed and compared to similar data recorded *in vivo*). Figure 2 illustrates the three most commonly observed spontaneous patterns of neural activity. These patterns appeared to be identical to *in vivo* recordings of neural activity that mediated rejection (Fig. 2A, D1) and ingestion (Fig. 2B, D2) behaviors and an intermediate pattern (Fig. 2C, D3) whose behavioral significance is unclear at this time.

Although *in vitro* preparations spontaneously manifest several different types of rhythmic neural activity, these different patterns were not expressed in equal numbers. Significantly more rejection-like patterns were expressed. Thus, the CPG in the buccal ganglia appears to biased toward generating rejection-like motor programs. It should be noted, however, that modulatory transmitters can 'reconfigure' the CPG and alter the frequency and types of patterns that are expressed (see below).

The types of patterned neural activity that were elicited by electrical stimulation of several input pathways to the CPG was also examined. Non-specific stimulation, either brief trains of stimuli or long-duration tonic stimulation, of peripheral nerves elicited a mixtures of patterned patterns of bursting activity that was identical to the spontaneously occurring patterns (e.g., Fig. 2). Stimulation of different input pathways, however, appeared to selectively activate different functional aspects of the CPG. For example, stimulation of either the radula nerve (R n.) or the esophageal nerve (E n.) most often elicited rejection-like patterns, whereas stimulation of nerve 2 elicited more ingestion-like patterns.

Taken together, the above data indicate that: 1) the isolated buccal ganglia retains the circuitry necessary to generate behaviorally relevant, rhythmic neural activity; 2) this rhythmic activity is not simply periodic in nature, but rather is a complex, bursting pattern; 3) the neural circuit is multifunctional and switches between expressing several different patterns; and 4) different functional configurations of the CPG can be selectively activated by input pathways. Given the ability to record intracellularly from identified cells in the buccal ganglia preparation (see below), this preparation affords an excellent model system in which to analyze the mechanisms underlying the generation of complex rhythms.

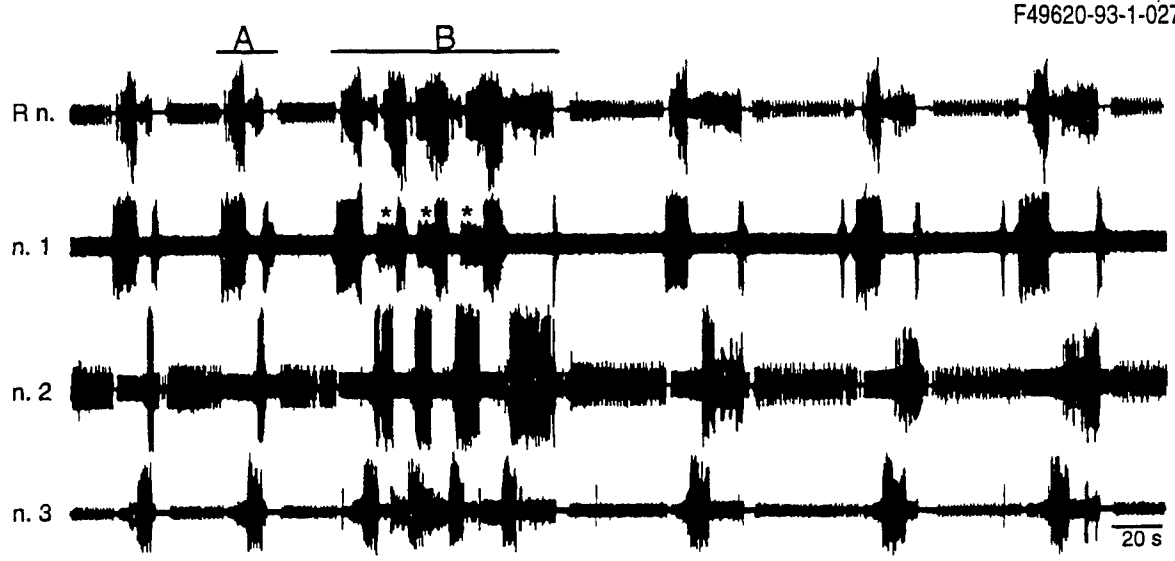

Fig. 1. Extracellular Recordings of Rhythmic Neural Activity from an Isolated Buccal Ganglia. The buccal ganglia were perfused with control saline and spontaneous neural activity was recorded extracellularly from the radula nerve (R n.) and buccal nerves 1, 2 and 3 (n.1, n.2 and n.3, respectively). Ordered bursts of large-unit activity were observed in all four buccal nerves. These data illustrate that the rhythmic activity in the buccal ganglia is not simply periodic in nature, but manifest complex, bursting activity. Rhythmic activity most often occurred as discreet, individual patterns (e.g., the neural activity by bar labeled A), but occasionally the rhythmic activity occurred as a 'chain' of multiple cycles of bursting activity (e.g., the neural activity indicated by the bar labeled B). Although these 'chains' had many features similar to those observed in the individual patterns, one difference that was constantly observed was a burst of medium-unit activity in nerve 1 (e.g., the bursts of medium-unit activity are indicated by the *).

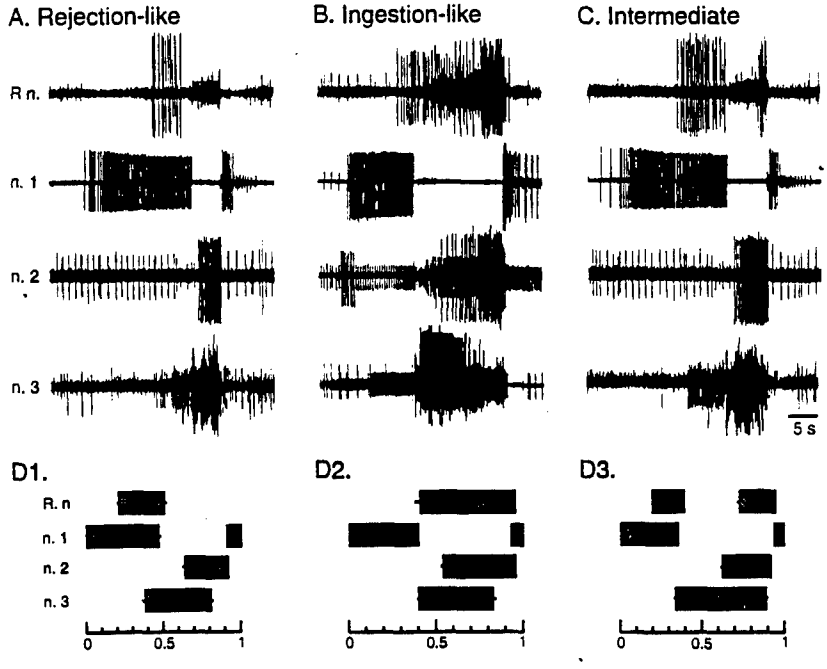

Fig. 2. The CPG in the Buccal Ganglia Expressed Several Distinct Patterns of Activity. The phase relationships among the bursts of activity in the four nerves were analyzed and compared to similar data recorded *in vivo*. At least three patterns were distinguished. One pattern (**A, D1**) was similar to that mediating rejection *in vivo*, another (**B, D2**) was similar to that mediating ingestion *in vivo*, and the third (**C, D3**) was an intermediate pattern whose behavioral relevance is not known. The horizontal axis in Panel D represents the normalized cycle length where 0 was defined as the beginning of activity in n. 1 and 1 as the end of large-unit activity in n. 1. During the rejection-like pattern, large-unit activity in the radula nerve (R n) occurred prior to large-unit activity in nerve 2 (n. 2), whereas during the ingestion-like pattern, there was substantial overlap of large-unit activities in R n. and n. 2. During the intermediate-like motor program, two bursts of large-unit activity were recorded in R n.; the first burst occurred prior to large-unit activity in n. 2 and a second burst overlapped with large-unit activity in n. 2. These data suggest that the CPG in the buccal ganglia is multifunctional.

B. Identification of Additional Cellular and Synaptic Elements of the CPG

A prerequisite to investigating the mechanisms underlying the rhythmic activity in the buccal ganglia is the identification of the cells and synaptic connections mediating the activity. Previous work in this laboratory and by others identified a number of cells that were believed to function as a CPG (e.g., B4/5, B31/32, B35, and B51). In addition, during the previous funding period, the synaptic connections among these cells was characterized (Fig. 3A) as were the phase relationships among bursts of action potentials in these cells during rhythmic activity. Intracellular recordings indicated that cells B31/32, B35 and B52 all fire during the protraction phase (P-phase) of the pacemaker activity, whereas B4/5 fired during the retraction phase (R-phase) (Fig. 4A). Hyperpolarizing either B31, B32 or B35 blocked rhythmic activity. Conversely depolarizing any one of these cells initiated rhythmic activity. Thus, cells B31/32 and B35 appeared to be crucial elements of the CPG.

To investigate whether the neural network illustrated in Fig. 3A could function as a CPG, a computational model of the circuit was developed. Simulations indicated that the circuit illustrated in Fig. 3A could not generate rhythmic activity. Moreover, these simulations predicted the properties and synaptic connections of an additional cell that, when incorporated into the network, would allow for the generation of rhythmic activity. Guided by these predictions, this laboratory and others identified a cell, B64, that manifests many of the properties predicted by the computational studies. For example, B64 was active during the R-phase (Fig. 4B), it inhibits P-phase neurons such as B31/32 and B52 and it excites R-phase neurons such as B4/5 and B51 (Fig. 3B). Moreover, in response to depolarizing current pulses, B64 fires spikes in all-or-nothing bursts. If B64 is hyperpolarized, then the R-phase is blocked. Thus, B64 appears to be the key element mediating the R-phase of pacemaker activity.

Studies during the current funding period indicated that dopamine modulates several aspects of rhythmic activity in the buccal ganglia (see below). For example, perfusing preparations with dopamine increased the frequency of rhythmic activity and biased the output of the CPG toward ingestion-like activity. Given the profound effects of dopamine on the functional configuration of the CPG, efforts were begun to identify putative dopamine-containing neurons in the buccal ganglia and characterize their actions. Histofluoresent studies in this laboratory and previous work by others found five large cell bodies in the buccal ganglia that had positive reactivity for catecholamines. These cells included a pair of medial cells, a pair of lateral cells and one unpaired cell. The medial pair have been recently characterized and are referred to as B20. Thus, studies in this laboratory focused on the lateral pair of catecholamine-containing neurons, which were refer to as B65. The synaptic connections of B65 are illustrated in Fig. 3B. The actions of B65 appeared to mimic many of the actions of exogenously applied dopamine, such as increasing the frequency of ongoing activity and biasing that activity toward ingestion-like patterns. Given the important role that dopamine plays in modulating rhythmic activity in the CPG (see below), B65 is likely to be a key element in the circuitry that controls the overall dynamics of the CPG.

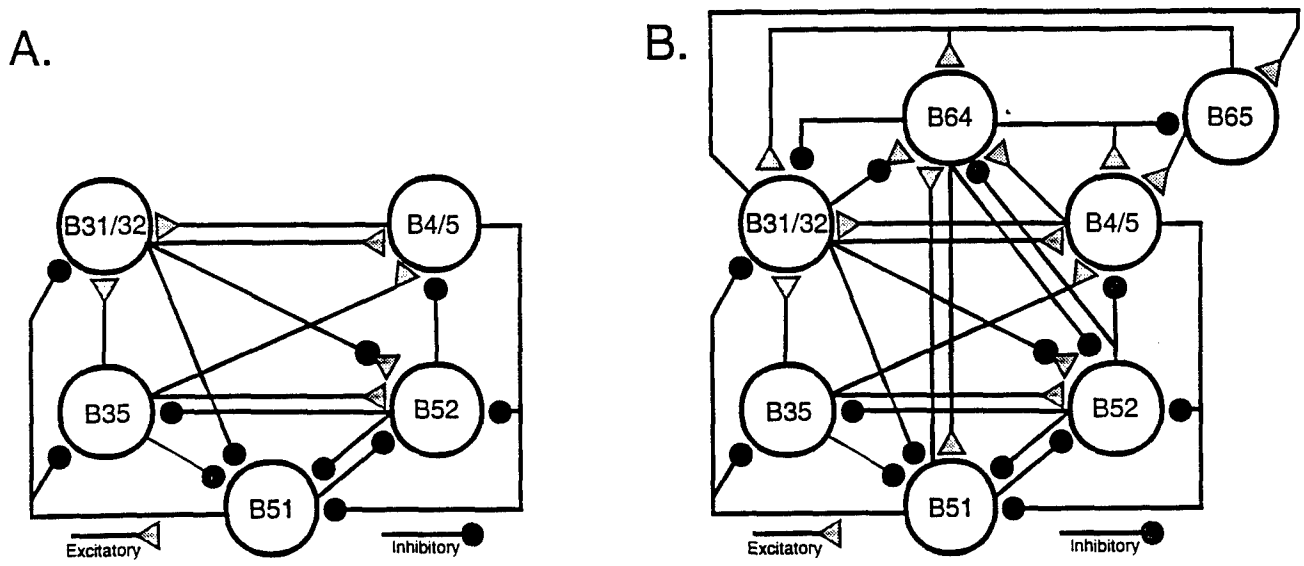

Fig. 3. Neural Circuitry Mediating Rhythmic Activity in the Buccal Ganglia. **A:** Previous studies identified several neurons (B4/5, B31/32, B35 and B51) that, when depolarized, elicited rhythmic activity in the buccal ganglia. During the previous funding period, the synaptic connections among these neurons were characterized. In addition, a computational model of this neural circuit was developed and its ability to generate rhythmic activity was examined. The simulations indicated that this circuit could not generate rhythmic activity. **B:** Using the predictions from the computational studies, neuron B64 was identified and many of its synaptic connections were characterized. Simulation studies indicated that this circuit can produce rhythmic patterns of activity. (Note, electrical coupling among cells is not indicated.)

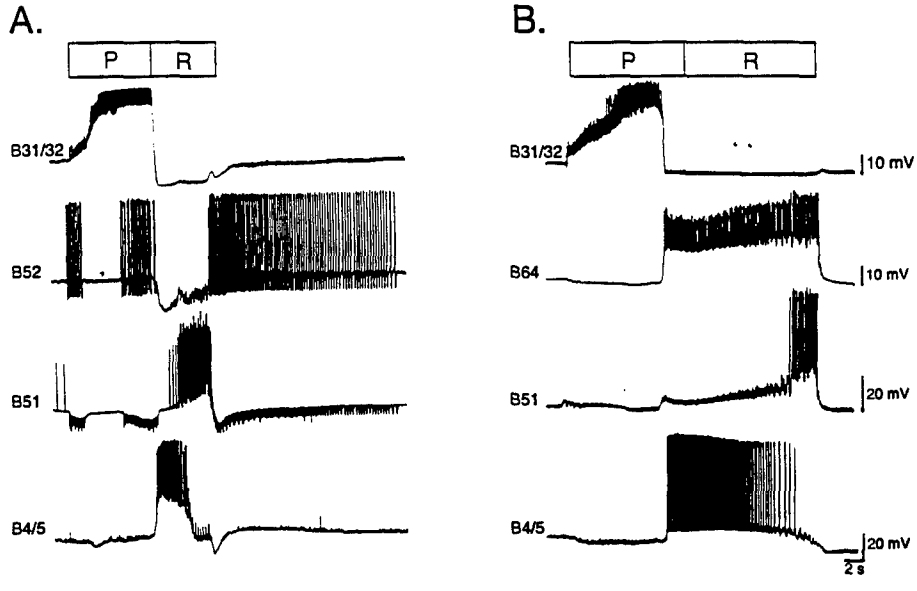

Fig. 4. Intracellular Recordings During Rhythmic Activity in the Buccal Ganglia. The pacemaker in the buccal ganglia generates two basic phases of activity: the protraction phase (P-phase) and the retraction phase (R-phase). This basic pacemaker activity, in turn, drives a number of distinct patterns (e.g., Fig. 2). **A:** Cells B31/32 are depolarized during the P-phase. The abrupt termination of this depolarization and a large inhibitory postsynaptic potential (IPSP) in B31/32 mark the transition from the P-phase to the R-phase. Experimentally, hyperpolarizing either B31 or B32 blocks rhythmic activity, which suggests that B31/32 are key elements mediating the P-phase. Although cell B52 has a high level of spontaneous activity, it also fires a burst during the P-phase and is inhibited during the R-phase. Cells B4/5 and B51 are depolarized and fire bursts of spikes during the R-phase. Although B51 makes an inhibitory connection to B31/32 (see Fig. 3), computer simulations indicated that this connection could not account for the transition from the P-phase to the R-phase. **B:** Guided by the predictions of the computer simulations, cell B64 was identified. B64 makes strong inhibitory connections to all cells that fire during the P-phase and strong excitatory connections to cells that fire during the R-phase. Indeed, if B64 is hyperpolarized, then the R-phase is blocked. Thus, B64 appears to be the key element mediating the R-phase of pacemaker activity.

C. Functional Plasticity of the CPG and its Rhythmic Activity

One of the salient features of CPGs is their ability to be modulated by extrinsic and intrinsic factors. Such modulation can play important roles in behavioral switching and/or regulation of a behavior by factors such as circadian rhythms or motivational states. The mechanisms by which this modulation occurs are poorly understood, however. The laboratory has begun to examine several issues related to functional plasticity in the CPG.

1. The actions of two transmitters, dopamine (DA) and serotonin (5HT) on rhythmic activity generated by the CPG were examined. Both of these agents have been implicated in controlling aspects of feeding behavior. For example, these transmitters are found in sensory pathways to the buccal ganglia and in higher-order neurons (e.g., command neurons and circadian pacemakers) that regulate feeding behavior.

DA had three effects. First, DA increased the frequency of spontaneously occurring motor programs. Second, DA increased the number of ingestion motor programs that were expressed and decreased the number of rejection motor programs. Third, in the presence of DA, stimulation of sensory pathways elicited ingestion motor programs, rather than a mix of both ingestion and rejection patterns. In contrast, 5HT decreased the frequency of spontaneously occurring motor programs, did not alter the relative distribution of the types of motor programs that were expressed, and in the presence of 5HT, stimulation of sensory pathways failed to elicit coherent patterns neural activity.

To further analyze the effects of these two neuromodulatory agents, a semi-intact preparation was developed and used to provide direct behavioral evidence correlating feeding behaviors with the actions of DA and 5HT. The preparation consisted of the isolated anterior portion of the animal (i.e., the head) with attached CNS. Feeding movements that were produced by the semi-intact preparation were monitored both with a force transducer and with a video-tape recorder. In control saline, feeding movements occurred spontaneously. These movements, however, were relatively weak, not well organized and occurred at very low frequencies. Perfusing the semi-intact preparation with DA significantly increased the frequency of feeding movements, enhanced the coordination of these movements and increased their force. These DA-induced movements were similar to biting in the intact animal. 5HT did not significantly change the frequency of feeding movements, but in the presence of 5HT, the force of the movements was increased and these movements appeared similar to swallowing in the intact animal.

These results indicate that biogenic amines play a role in determining the functional configuration of the CPG and its responsiveness to sensory inputs. In the semi-intact preparation, 5HT induced well organized swallowing movements. In contrast, 5HT appeared to disrupt or inhibit patterned neural activity in the isolated buccal ganglia. One possibility for the different actions of 5HT in the reduced vs. semi-intact preparations is that the actions of 5HT may arise from more complex integration by additional central and/or peripheral neural elements, which are not present in the reduced preparation. The actions of DA appeared similar in both preparations. In the isolated buccal ganglia, DA biased the output of the CPG toward ingestion-like neural activity. This DA-induced reconfiguration of the CPG, in turn, was manifested as coordinated biting movements in the semi-intact preparation.

2. The phase-dependent modulation of rhythmic activity was examined. A hallmark of biological rhythms is that they are generated endogenously and thus, are expressed in isolation

(i.e., free-running activity). Although the oscillations are self-sustaining, they can be modified by external signals (e.g., entrainment to periodic stimulation). When the external signals are removed, it is generally assumed that the oscillatory system will revert to its free-running pattern of activity. This need not be the case, however. Biological oscillators are often multifunctional and thus, in theory, appropriately timed stimuli could induced changes in the rhythmic activity that persist after the external stimuli have been removed.

We have begun to examine phase-dependent modulation of rhythmic activity in the CPG of the buccal ganglia. Rhythmic activity was elicited by tonic (i.e., non-specific) stimulation (2 Hz) of the buccal nerve 2,3. The rhythmic activity was a mixture of ingestion-like, rejection-like and intermediate patterns (see Fig. 2). The ongoing rhythmic activity was perturbed by brief, phasic stimulation of the esophageal nerve (E n.). Three groups of preparations were examined: contingent stimulation, yoke-control, control groups. In the contingent stimulation group, E n. stimulation applied immediately following each ingestion-like pattern. In the yoke-control group, the same type of E n. stimulation was used, but now this stimulation was not associated with any specific phase or type of rhythmic activity. In the control group, no E n. stimulation was used. After the E n. stimulation was terminated, a significant increase in the frequency of rhythmic activity was observed in the contingent stimulation group compared to either the yoke-control or the control groups. There was no significant difference between the yoke-control and the control groups. The modification of the rhythmic activity in the contingent stimulation group corresponded to a specific increase of the frequency of the ingestion-like pattern (i.e., the pattern that received the phase-specific E n. stimulation). No significant difference in the frequency of either rejection-like or intermediate patterns was observed.

These results indicate that the effects of an input pathway on the pacemaker can be phase-dependent and that the induced modifications to the pacemaker can persist in the absence of any addition stimuli. These phase-dependent effects add a level of complexity to potential control processes of pacemaker activity.

It is also intriguing to note the striking similarity between phase-dependent modulation of rhythmic activity in a pacemaker and an important form of associative learning, operant conditioning. In an operant conditioning paradigm, the delivery of a reinforcing stimulus is made contingent upon the expression of a designated behavior (i.e., the operant). As a result of the contingent reinforcement, the frequency at which the designated behavior is expressed increases, in much same manner that phase-specific stimuli increase the of the expression of ingestion-like patterns. Thus, this preparation may afford an opportunity to gain insights into the cellular mechanisms underlying operant conditioning as well as mechanisms of biological rhythms.

3. The influence of motivational states on properties of the oscillator was examined. Motivational states are inferred mechanisms that direct voluntary behavior. Although motivational states play a key role in behavior, the physiological manifestations of motivational states are not well understood. During the project period, this issue was investigated by determining whether motivational states can alter the properties of the CPG that mediates feeding behavior.

Previous studies have indicated that feeding behavior of *Aplysia* is influenced by several motivational states, such as arousal and satiety. Since satiation is manifested as a cessation of ingestive feeding movements, it is possible that satiation influences the response of the CPG to DA, which normally biases the output of the CPG toward ingestion (see above). To test this possibility, animals were food deprived for 5-6 days and then were either fed to satiation or not,

and motor programs were monitored in buccal ganglia that were isolated from these two groups of animals. In food deprived animals, DA increased the frequency of spontaneously occurring motor programs and biased this neural activity towards ingestion motor programs. In satiated animals, DA did not change the frequency of motor programs nor did DA bias the neural activity towards ingestion.

These results indicate that the CPG can exist in more than one stable configuration (i.e., one configuration that is responsive to DA and another that is not) and that the functional organization of this CPG is reflective of the motivational state of the animal. In addition, these results suggest that changes in behavior that accompany different motivational states do not require sustained inputs from either sensory or higher-order neurons, since changes in motor programs were detected in the isolated preparation.

D. Summary and conclusions

Several general principles are emerging from these studies. First, the functional organization of neural circuits is dynamic, and a single circuit, such as a CPG, can produce several distinct outputs, which in turn, can mediate different behaviors. Second, modulatory transmitters can regulate the functional organization of circuits as well as their responsiveness to inputs. Third, motivational systems can influence behaviors, in part, by acting on motor systems, such as CPGs. Fourth, motor systems possess cellular mechanisms capable of supporting complex forms of neuronal plasticity, which in turn, may contribute to learning and memory. These general principles illustrate that motor behaviors are governed by highly adaptive neural networks and help to explain how systems of nerve cells function to produce and modulate behavior.

IV. Personnel Supported and/or Associated with the Research Effort

Andrew Adams	Undergraduate
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Kara Herynk	Research Assistant
Wenyao Jia	Computer Programmer
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Joe Kong	Computer Programmer
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Romuald Nargeot, Ph.D.	Postdoctoral Research Fellow
Gary Patterson	Undergraduate/Research Assistant
Andrew Tang	Undergraduate
Chun Wang	Undergraduate
Bruce Welch	Electronic Eng.
Hongqin Zhang	Undergraduate
Israel Ziv, Ph.D.	Research Consultant

V. Publications

A. Abstracts

1. Kabotyanski, E.A., Baxter, D.A. and Byrne, J.H. 1994. Identification of a catecholaminergic neuron in the buccal ganglia of *Aplysia* that initiates rhythmic neuronal activity indicative of ingestion. *Soc. Neurosci. Abstr.* 20: 23.
2. Byrne, J., 1995. Neuronal and network determinants of simple and higher-order features of associative learning: experimental and modeling approaches. *Proceedings of the 23rd Gottingen Neurobiology Conference*, eds., Elsner, N. and Menzel, R., Stuttgart, Georg Thieme Verlag, p. 66.
3. Cushman, S.J., Baxter, D.A. and Byrne, J.H. 1995. Central representation of satiation of feeding behavior in *Aplysia*. *Soc. Neurosci. Abstr.*, 21: 151.
4. Baxter, D.A., Cushman, S.J., Kabotyanski, E.A. and Byrne, J.H. 1995. Characterization of motor programs generated in isolated buccal ganglia of *Aplysia* and their modulation by biogenic amines. *Soc. Neurosci. Abstr., 21*: 1766.
5. Kabotyanski, E.A., Baxter, D.A. and Byrne, J.H. 1995. Dopamine and serotonin coordinate different aspects of consummatory feeding behavior in *Aplysia*. *Soc. Neurosci. Abstr.*, 21: 1766.
6. Nargeot, R., Baxter, D.A. and Byrne, J.H. 1995. Afferent control of buccal motor programs in *Aplysia*. *Soc. Neurosci. Abstr.*, 21: 1766.
7. Nargeot, R., Baxter, D.A. and Byrne, J.H. 1996. Neural analogue of operant conditioning of feeding behavior in *Aplysia*. *Soc. Neurosci. Abstr.*, 22: 1438.

B. Articles and Chapters

1. Kabotyanski, E.A., Ziv, I. Baxter, D.A. and Byrne, J.H. 1994. Experimental and computational analyses of a central pattern generator underlying aspects of feeding behavior in *Aplysia*. *Netherlands J. Zool.* 44: 357-373.
2. Byrne, J.H. and Crow, T. 1995. Invertebrate models of learning: *Aplysia* and *Hermissenda*. In: *Handbook of Brain Theory and Neural Networks*, ed., M. Arbib, MIT Press/Bradford Books, pp. 487-491.
3. Byrne, J.H., Sugita, S. and Baxter, D.A. 1995. Roles of multiple second messenger systems in the serotonergic modulation of spike duration, membrane currents and synaptic connections a *Aplysia* sensory neurons. In: *Basic Neuroscience of Invertebrates*, eds., Koike, H., Takahashi, K. and Kidokore, Y. Business Center for Academic Societies Japan, pp. 93-109.
4. Byrne, J.H. and Kandel, E.R. 1996. Presynaptic facilitation revisited: State- and time-dependence. *J. Neurosci.*, 16: 425-435.
5. Baxter, D.A., Canavier, C.C. Lechner, H., Butera, R.J., DeFranceschi, A.A., Clark, J.W. and Byrne, J.H. 1996. Coexisting stable oscillatory states in single cell and multicellular neuronal oscillators. In: Levine, D., Brown, V. and Shirey, T. (Eds.), *Oscillations in Neural Systems*, Hillsdale: Lawrence Erlbaum and Associates, in press.

C. Manuscripts in Preparation or Under Review

1. Nargeot, R., Baxter, D.A. and Byrne, J.H. Contingent reinforcement of motor programs: a neural analogue of operant conditioning. (In preparation to be submitted to *J. Neurosci.*)
2. Kabotyanski, E.A., Baxter, D.A. and Byrne, J.H. Identification and characterization of catecholaminergic neuron B65 that initiates patterned activity in the buccal ganglia of *Aplysia.* (In preparation, to be submitted to *J. Neurophysiol.*)
3. Baxter, D.A., Cushman, S.J., Kabotyanski, E.A. and Byrne, J.H. Patterns of neural activity generated in isolated buccal ganglia of *Aplysia.* I. Characterization of feeding motor programs and their modulation by dopamine and serotonin. (In preparation to be submitted to *J. Neurophysiol.*)
4. Baxter, D.A. and Byrne, J.H. Patterns of neural activity generated in isolated buccal ganglia of *Aplysia.* II. Synaptic interactions among neurons that are believed to function as a central pattern generator. (In preparation, to be submitted to *J. Neurophysiol.*)
5. Lechner, H.E., Baxter, D.A. and Byrne, J.H. 1996. Appetitive classical conditioning of consummatory feeding behavior in *Aplysia.* (In preparation, to be submitted to *Behav. Neurosci.*)

VI. Interactions/Transitions

A. Participation/Presentations at Meeting, Conferences, Seminars

1. Baxter, D.A and Byrne, J.H. Serotonin inhibits rhythmic neural activity mediated via neurons B31/32 in the buccal ganglia of *Aplysia.* 23rd Annual Meeting of the Society for Neuroscience, Washington D.C., November, 1993.
2. Baxter, D.A., Ziv, I. and Byrne, J.H. Simulator for neural networks and action potentials (SNNAP): use of computer simulations as a supplement for undergraduate and graduate courses in neurobiology. 23rd Annual Meeting of the Society for Neuroscience, Washington D.C., November, 1993.
3. Kabotyanski, E.A., Ziv, I., Baxter, D.A. and Byrne, J.H. Metabolic precursors of dopamine and serotonin modulate rhythmic neural activity in the buccal ganglia of *Aplysia.* 23rd Annual Meeting of the Society for Neuroscience, Washington D.C., November, 1993
4. Baxter, D.A. and Byrne, J.H. Simulating features of associative learning in single cells and small networks. 1st World Congress on Computational Medicine, Public Health and Biotechnology, Austin, TX, April, 1994.
5. Kabotyanski, E.A., Baxter, D.A. and Byrne, J.H. Identification and characterized of B65: a catecholaminergic neuron in the buccal ganglia of *Aplysia.* 4th Conference on Simpler Nervous Systems, Moscow, Russia, May, 1994.
6. Kabotyanski, E.A., Baxter, D.A. and Byrne, J.H. Experimental and computational analyses of a central pattern generator underlying aspects of feeding behavior in *Aplysia.* 4th Symposium on Molluscan Neurobiology (SYMON IV), Amsterdam, Netherlands, June, 1994.

7. Baxter, D.A., Ziv, I. and Byrne, J.H. Simulator for neural networks and action potentials (SNNAP). 4th Symposium on Molluscan Neurobiology (SYMON IV), Amsterdam, Netherlands, June, 1994.
8. Byrne, J.H. Roles of multiple second messenger systems in the serotongeric modulation of spike duration and membrane currents in *Aplysia* sensory neurons. International Symposium in Basic Neuroscience in Invertebrates, Tokyo, Japan, Oct. 1994.
9. Kabotyanski, E.A., Baxter, D.A. and Byrne, J.H. Identification of a catecholaminergic neuron in the buccal ganglia of *Aplysia* that initiates rhythmic neuronal activity indicative of ingestion. 24th Annual Meeting of the Society for Neuroscience, Miami, FL, November, 1994.
10. Kabotyanski, E.A., Baxter, D.A. and Byrne, J.H. Identification of a catecholaminergic neuron in the buccal ganglia of *Aplysia* that initiates rhythmic neuronal activity indicative of ingestion. 13th Annual Houston Conference on Biomedical Engineering Research, Houston, TX, February, 1995.
11. Cushman, S.J., Baxter, D.A., Kabotyanski, E.A. and Byrne, J.H. Modulation of buccal motor programs in *Aplysia* by 5HT and dopamine. 13th Annual Houston Conference on Biomedical Engineering Research, Houston, TX, February, 1995.
12. Cushman, S.J., Baxter, D.A. and Byrne, J.H. Central representation of satiation of feeding behavior in *Aplysia*. 21st Annual Meeting of the Society for Neuroscience, San Diego, CA, November, 1995.
13. Baxter, D.A., Cushman, S.J., Kabotyanski, E.A. and Byrne, J.H. Characterization of motor programs generated in isolated buccal ganglia of *Aplysia* and their modulation by biogenic amines. 21st Annual Meeting of the Society for Neuroscience, San Diego, CA, November, 1995.
14. Kabotyanski, E.A., Baxter, D.A. and Byrne, J.H. Dopamine and serotonin coordinate different aspects of consummatory feeding behavior in *Aplysia*. 21st Annual Meeting of the Society for Neuroscience, San Diego, CA, November, 1995.
15. Nargeot, R., Baxter, D.A. and Byrne, J.H. Afferent control of buccal motor programs in *Aplysia*. 21st Annual Meeting of the Society for Neuroscience, San Diego, CA, November, 1995.
16. Dr. Byrne was an invited speaker at the 23rd Gottingen Neurobiology Conference, 1995.
17. Dr. Byrne was an invited speaker at the New York University Symposium on Memory and Brain, New York, NY, 1995.
18. Nargeot, R., Baxter, D.A. and Byrne, J.H. Neural analogue of operant conditioning. 14th Annual Houston Conference on Biomedical Engineering Research, Houston, TX, 1996.

B. Consultative and Advisory Functions

1. Dr. Byrne

1993-present	Member of the Editorial Board of the journal *Learning and Memory*
1994-present	Assistant Editor for the journal *News in Physiological Sciences*
1994-present	Member of the Editorial Board of the journal *Behavioral Neuroscience*

1994-present Member of the Selection Committee for the Society for Neuroscience Young Investigator Award

1994-present Member of the Advisory Committee, John Sealy Memorial Endowment Fund for Biomedical Research

1996-present Editor of the journal *Learning and Memory*,

Conference co-organizer for a symposium on Learning and Memory, Cold Spring Harbor Laboratory, NY, November,1994.

Juror on the dissertation committee of Dr. R. Nargeot. Dr. Nargeot was a graduate student in the laboratory of Dr. M. Moulins at the University of Bordeaux and CNRS (France). The dissertation was entitled "Plasticity of stomatogastric neural networks of *Crustracea*: electrophysiological and pharmacological studies" and the Ph.D. was awarded in February, 1995.

Member of the Nominating Committee for officers for the AAAS Section of Neuroscience, 1995.

Member of the Outside Review Committee, Columbia University NIMH Program Project, 1995.

Member of the National Institute of Neurological Disorders and Stroke Special Review Committee on Conferences, 1995.

Outside reviewer for the International Human Frontier Science Program.

2. Dr. Baxter

Conference co-organizer for the 13th Annual Houston Conference on Biomedical Engineering Research, Houston, TX, February, 1995.

Outside reviewer for: National Science Foundation, New Zealand Neurology Foundation, Medical Research Council of Canada, *Journal of Neuroscience* and *Journal of Neurophysiology*.

C. Transitions

During a previous period of support from the AFOSR (Grant 91-0027), a general-purpose Simulator for Neural Networks and Action Potentials (SNNAP) was developed. The continued development and distribution of SNNAP is now supported by a grant from the National Center for Research Resources (NCRR Grant R01-RR11626). Additional information about SNNAP is current available via an internet homepage (http://nba19.med.uth.tmc.edu/~snnap/).

VII. New Discoveries, Inventions, or Patent Disclosures

none

VIII. Honors/Awards

John H. Byrne: NIMH Research Scientist Award